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L3: Entry 1 of 596 File: USPT Feb 6, 2001

DOCUMENT-IDENTIFIER: US 6183988 B1
TITLE: Leukocyte-specific protein and gene, and methods of use thereof

DEPR:
Sp140 and viral infection. IFN treatment increases the expression of each of the previously identified components of the NB (Sp100, PML and NDP52). This IFN induction of NB-associated proteins may have functional significance. For example, Sp100 may be involved in establishing an "anti-viral state" (Guldner, H. H. et al., J. Immunol. 149:4067-4073 (1992)), and it has recently been demonstrated that overexpression of PML in Hep2 cells dramatically decreases the ability of these cells to support adenoviral replication (Doucas, V. et al., Genes & Devel. 10:196-207 (1996)). Based on these results, it has been hypothesized that increased expression of PML may delay or block Sp100 and NDP52 recruitment to viral replication domains resulting in inhibition of viral DNA replication (Doucas, V. et al., Genes & Devel. 10:196-207 (1996)). The identification in the present invention of Sp140, a novel NB component that is expressed in cells involved in host defense, raises the possibility that Sp140, like other NB components, may have a role in the pathogenesis of, or the host response to, viral infection.

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L3: Entry 2 of 596 File: USPT Feb 6, 2001

DOCUMENT-IDENTIFIER: US 6183751 B1
TITLE: Unique associated Kaposi's Sarcoma virus sequences and uses thereof

DEPR:
This invention provides the isolated KSHV polypeptide comprising DNA polymerase encoded by ORF 9. In one embodiment, DNA polymerase comprises an enzyme essential for viral replication, inhibition of which prevents virus production. In another embodiment, DNA polymerase comprises a subunit vaccine. In another embodiment, DNA polymerase comprises an antigen for immunologic assays.

DEPR:
The antiherpesvirus agents that will be useful for treating virus-induced KS can be grouped into broad classes based on their presumed modes of action. These classes include agents that act (1) by inhibition of viral DNA polymerase, (2) by targeting other viral enzymes and proteins, (3) by miscellaneous or incompletely understood mechanisms, or (4) by binding a target nucleic acid (i.e., inhibitory nucleic acid therapeutics, supra). Antiviral agents may also be used in combination (i.e., together or sequentially) to achieve synergistic or additive effects or other benefits.

DEPR:
Many antiherpesvirus agents in clinical use or in development today are nucleoside analogs believed to act through inhibition of viral DNA replication, especially through inhibition of viral DNA polymerase. These nucleoside analogs act as alternative substrates for the viral DNA polymerase or as competitive inhibitors of DNA polymerase substrates. Usually these agents are preferentially phosphorylated by viral thymidine kinase (TK), if one is present, and/or have higher affinity for viral DNA polymerase than for the cellular DNA polymerases, resulting in selective antiviral activity. Where a nucleoside analogue is incorporated into the viral DNA, viral activity or reproduction may be affected in a variety of ways. For example, the analogue may act as a chain terminator, cause increased lability (e.g., susceptibility to breakage) of analogue-containing DNA, and/or impair the ability of the substituted DNA to act as template for transcription or replication (see, e.g., Balzarini et al., supra).

DEPR:
Other useful antiviral agents include:

5-thien-2-yl-2'-deoxyuridine derivatives, e.g., BTDU [5-5(5-bromothien-2-yl)-2'-deoxyuridine] and CTDU [b-(5-chlorothien-2-yl)-2'-deoxyuridine]; and OXT-A [9-(2-deoxy-2-hydroxymethyl-.beta.-D-erythro-oxetanosyl)adenine] and OXT-G [9-(2-deoxy-2-hydroxymethyl-.beta.-D-erythro-oxetanosyl)guanine]. Although OXT-G is believed to act by inhibiting viral DNA synthesis its mechanism of action has not yet been elucidated. These and other compounds are described in Andrei et al., 1992, Eur. J. Clin. Microbiol. Infect. Dis. 11, 143-51. Additional antiviral purine derivatives useful in treating herpesvirus infections are disclosed in U.S. Pat. No. 5,108,994 (assigned to Beecham Group P.L.C.). 6-Methoxypurine arabinoside (ara-M; Burroughs Wellcome) is a potent inhibitor of varicella-zoster virus, and will be useful for treatment of KS.

DEPR:
Although applicants do not intend to be bound by a particular mechanism of antiviral action, the antiherpes-virus agents described above are believed to act through inhibition of viral DNA polymerase. However, viral replication requires not only the replication of the viral nucleic acid but also the production of viral proteins and other essential components. Accordingly, the present invention contemplates treatment of KS by the inhibition of viral proliferation by targeting viral proteins other than DNA polymerase (e.g., by inhibition of their synthesis or activity, or destruction of viral proteins after their synthesis). For example, administration of agents that inhibit a viral serine protease, e.g., such as one important in development of the viral capsid will be useful in treatment of viral induced KS.

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L3: Entry 3 of 596 File: USPT Jan 23, 2001

DOCUMENT-IDENTIFIER: US 6177272 B1
TITLE: Method for treating vascular proliferative diseases with p27 and fusions thereof

BSPR:
The HSV-1 thyrnidine kinase gene (TK) is the most widely used suicide gene in mammalian systems. TK efficiently phosphorylates guanosine analogs ganciclovir (GCV) and acyclovir (ACV), which are subsequently phosphorylated by cellular enzymes into their triphosphate forms. These end-products are incorporated into the growing DNA chain, leading to elongation arrest (ACV) or a drastic slow down in DNA synthesis (GCV). Death usually ensues, through a mechanism identified in some cell lines as apoptosis. The mechanism that triggers cell death is not known. In the case of GCV, another action other than at the level of the DNA polymerase inhibition might exist, since no correlation is observed between the inhibition of mutant viral DNA polymerase by GCV and growth of these mutants in the presence of GCV.

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L13: Entry 3 of 1761 File: USPT Feb 6, 2001

DOCUMENT-IDENTIFIER: US 6184355 B1
TITLE: FAE1 genes and their uses

BSPR:
A "polynucleotide sequence from an FAE1 gene" is a subsequence or full length polynucleotide sequence of an FAE1 gene which, when present in a transgenic plant, has the desired effect, for example, inhibiting expression of the endogenous FAE1 gene. In the case of both expression of transgenes and inhibition of endogenous genes (e.g., by antisense, or sense suppression) one of skill will recognize that the inserted polynucleotide sequence need not be identical and may be "substantially identical" to a sequence of the gene from which it was derived. As explained below, these variants are specifically covered by this term.

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L15: Entry 1 of 70 File: USPT Feb 6, 2001

DOCUMENT-IDENTIFIER: US 6184369 B1
TITLE: Anti-viral guanosine-rich oligonucleotides

DRPR:
FIG. 7 shows continued suppression of HIV-1 p24 production seven days post removal of oligonucleotide. Four days post-infection with HIV-1.sub.DV, the media from infected cells treated with oligonucleotides (2.5 .mu.M) was removed and replaced with fresh media without oligonucleotide. The presence of viral p24 antigen was then assayed 7 and 11-days post infection. All samples were assayed in quadruplicate and the average values used to plot this graph. The legend to the right of the graph indicates the symbol used for each oligonucleotide tested.

DEPR:
The anti-HIV-1 activity of a series of GTOs, with PD backbones, containing different sequences motifs was tested. One of the sequence motifs tested (oligonucleotide I100-07) was 10 fold more active at inhibiting HIV-1 induced syncytium formation than the other motifs tested (e.g. I100-00 shown in Table 1). I100-07 and its derivatives (length and chemical modification) were further tested for their ability to inhibit virus in a dose-dependent fashion by measurement of syncytium formation and viral p24 production. Briefly, HIV-1.sub.DV was used to infect the SUP T1 lymphoblastoid cell line at a multiplicity of infection (m.o.i.) of 0.1 TCID.sub.50 for one hour at 37.degree. C. prior to washing and resuspension in increasing concentrations of GTOs. The cells (2.times.10.sup.4 cells/well) were inoculated in triplicate in 200 ul of RPMI 1640 containing 10% fetal calf serum. Four days later, the number of syncytia per well or the level of p24 in the medium was determined. The results of these assays are presented in Table 4.

DEPR:
The duration of the viral suppression was assayed by changing the medium in HIV-1 infected cultures containing 2.5 uM of various oligonucleotides to complete media without added oligonucleotide on day 4 post-viral infection. The production of viral p24 antigen was then assayed on day 7 and day 11 post-infection. The results of this experiment indicated that the shorter variants of I100-07 (I100-15 and I100-16) as well as the PT version of this molecule (I100-21), were capable of totally suppressing HIV-1 p24 production for at least 7 days after removal of the drug from the culture medium (Table 6). This substantial level of prolonged inhibition was >99% for I100-15, I100-16 and I100-21 when compared to the p24 antigen levels obtained for untreated HIV-1 infected cells (Table 6). The quantitation of p24 production

infected cells (Table 6). The quantitation of p24 production relative to untreated HIV-1 infected SUP T1 cells for all oligonucleotides tested is presented in Table 6. The presence of sulfur molecules in the backbone of oligonucleotide I100-07 (I100-21) had a more marked effect on the reduction of virus seven days after removal of compound from the culture medium than was observed at the four day post-infection assay point (Table 5).

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